REVIEWS.

ART. XXII.—*Études Expérimentales et Cliniques sur les Traumatismes Cérébraux.* Par le Dr. H. DURET. Tome I., 8vo., pp. 327, XIX planches. Paris: Le Progrès Médical and A. Delahaye, 1878.

Experimental and Clinical Studies upon Cerebral Traumatisms. By Dr. H. DURET.

THE importance of the brain as an intellectual and vital centre, the obscurity and complexity of its lesions, and the difficulties and uncertainties of their treatment have made, and always will make, it an object of the greatest (we had almost said melancholy) interest to both physicians and surgeons, and this interest has been much quickened of late by the recent advances made in our knowledge of the special functions pertaining to different portions of the cerebral hemispheres, and by the success obtained by two French surgeons in cases of operative interference based upon this knowledge. The uncertainty concerning the exact seat of the lesion, especially when the original traumatism had not caused a recognizable fracture, and the symptoms had supervened tardily, made surgeons loath to interfere lest they should needlessly compromise their art and themselves, and the habit of hesitation thus induced led them, illogically, perhaps, but not unnaturally, to extend expectant treatment to other cases also in which more active measures were plainly called for, and even the point for the application of the trephine clearly indicated. When at last the urgency of the symptoms became so great that the surgeon was in a measure forced to operate, the condition of the patient had been rendered so grave by the inflammatory and destructive processes that had been allowed to develop, as to almost preclude recovery, and thus was established a vicious circle —delay in interference leading to fatal results, and the sequence of fatal results justifying or at least favouring delay—which has made trephining a rare operation, and reduced the surgeon almost to the position of a simple spectator in cases of injury to the skull. In some hospitals it is considered almost an axiom that the application of the trephine will be followed by the death of the patient, and in the recent discussions in the Académie de Médecine and the Société de Chirurgie upon the therapeutical uses to be made of the newly-acquired knowledge of motor centres, the great danger of this operation was the argument most strongly urged against interference when the interior of the cranium was not already exposed to the air.

It seems not unlikely that this fear of the trephine is a mischievous tradition handed down from generation to generation and as ill-founded as that of the intolerance of the urinary bladder so recently overthrown by Prof. Bigelow. Even if it is not so, antiseptic surgery has reduced, if it has not entirely removed, the danger, and the only thing that is now required to justify the habitual substitution of active interference for the present policy of expectation or resignation is greater certainty in the means of diagnosis.

For this reason we welcome any work that adds to our knowledge of the nature and the means of diagnosis of cerebral lesions; and it is to this task that the author of the book now before us has addressed himself. The present volume deals only with the experimental study of cerebral traumatisms, the clinical aspects will be considered in the 2d part, which is yet to appear. In this volume we have an analytical study of the pathology and symptomatology of primary lesions, of cerebral commotion, compression, and contusion, and we are promised in the second the synthetical application of the results here acquired to the clinical aspects of these lesions as they are encountered by the surgeon. The scope of this part is indicated by some of the titles of its chapters: cerebro-spinal shock, the influence of an excess of pressure, the mechanism of compression, and the part taken by the dura mater, the meningeal vessels, and the different portions of the encephalon in cerebral traumatisms. The most original, and in some respects the most important, part is that relating to the subject first mentioned, and nearly half the volume is given to its consideration.

Cerebro-spinal shock (choc cephalo-rachidien) is the term employed by M. Duret to indicate the sudden arrest or suppression of the functional activity of the brain produced by a blow upon the skull or by an injection into its cavity, when the force is transmitted through and by the cerebro-spinal liquid to certain more or less distant regions of the encephalon, notably the 4th ventricle, and there exerts a vulnerant action. The nature of the shock, the mode in which the action is exerted, and the lesions which it produces were, he claims, discovered by him and first described in the book before us. To the exposition of its nature, cause, and effects, he brings an enthusiasm which is fully justified by the importance of the discovery, and if we feel some hesitation in accepting his analysis of mechanical problems whose very factors are often hypothetical and always complex, we must admit that the opinions of a conscientious worker thoroughly imbued with the scientific spirit, as M. Duret unquestionably is, and applying the test of experiment whenever possible, are not to be lightly questioned by the critic who can give perhaps only minutes or hours to the consideration of questions that have occupied the author for days or weeks. And yet when we remember that even simple problems in physics are beset with difficulties arising from failure to apprehend or rightly estimate the mutual relations of the parts, we may well doubt whether any one is qualified to deal with those presented by the infinite complexity of the skull and its contents. But while we cannot follow M. Duret through all his elaborate and painstaking explanations of the mechanism by which certain effects are produced, we can accept heartily his demonstration of those effects and of their relation to well-known clinical symptoms, and instead of losing by the elimination may even gain by increase of sharpness of outline and precision of conception. We can only gain by fixing our attention upon experimentally-ascertained facts to the exclusion of inferential explanations of them, and by declining to follow the author into hypothetical vacuums produced by questionable *cônes de soulèvement.*

Two facts, two lesions stand out prominently in the record of the experiments; with one of them we are all in a measure familiar, with the discovery of the other and of the agency of the cerebro-spinal liquid in producing both M. Duret is to be credited. But we cannot do better than follow the author's own dramatic method and begin with "*How I discovered the cerebro-spinal shock.*"

He was seeking for the causes and the mode of production of concussion of the brain (*commotion cérèbrale*), and had succeeded several times in producing its clinical symptoms by rapidly and forcibly injecting non-absorbable liquids into the anterior of the skull, but in these and even in other cases where the injection of from ten to twenty grammes of water had killed dogs instantaneously, he had found no lesion sufficient to account for the result. The tetanic contraction of all the muscles, the arrest of respiration, and the slowing of the heart's action made him suspect a lesion of the medulla, and with the hope of making this lesion extensive enough to be recognized, he determined to inject a much larger quantity of water than he had previously employed in the experiments. 100 grammes (℥ 3 +) were injected rapidly by means of a syringe screwed into a small hole made near the centre of the right side of the skull. At the autopsy an extensive rent was found in the median line of the floor of the 4th ventricle, a rent evidently produced by a force acting from within outwards, and the aqueduct of Sylvius and the central canal of the cord were largely dilated.

When we recall the definite plan of the experiment, and note that the dog thus sacrificed was "no. 32 *bis*," the exultation which M. Duret manifests in the next paragraph seems quite as justifiable as that which sent Archimedes, naked, through the streets of Syracuse, and gave all languages a common cry of triumph. The mode in which the lesion had been produced was apparent: the pressure upon the surface of the hemispheres had forced the cerebro-spinal liquid contained within the lateral ventricles out through the only channel open to it, the aqueduct of Sylvius, into the 4th ventricle, and the latter, not being able to discharge the liquid through the minute foramen of Magendie and the central canal of the spinal cord as rapidly as it was received, was subjected to a dilating pressure sufficient to rupture its walls. In order to eliminate a possible source of error, the direct passage of the injected liquid into the ventricle, the syringe was filled with a thick solution of gelatine instead of water, and the experiment was repeated. The result was the same: muscular rigidity, arrest of respiration, death; and at the autopsy the coagulated gelatine was found solely between the dura mater and the bone. There was a small amount of blood in the cavity of the arachnoid at the base of the brain, the aqueduct of Sylvius was enormously dilated and torn, the central canal of the cord was dilated throughout its entire length, and the floor of the 4th ventricle and the restiform bodies were covered with hemorrhagic spots.

It is too evident to escape the notice of any one that an experiment of this kind, however interesting and valuable it may be from some points of view, cannot be considered an experimental reproduction of any traumatism inflicted upon the skull. The amount of the injected liquid was so great that the compression exerted by it was far beyond that which could be produced by any blow unaccompanied by a crushing fracture. It could serve only to show the manner in which and the point at which a certain much less severe effect might be produced. Granted that the sudden passage of the contents of the lateral ventricles into the fourth ventricle may cause rupture of the latter, it remains to be shown whether a blow upon the skull can cause this passage, and, if it can, by what means; and here we must reverse the Cartesian order followed in this instance by M. Duret, and show what the blow actually does before we try to show what it ought to do, and how it ought to do it. It is not difficult to find material for our purpose; the book is filled with records of experiments, and we take one, almost at random, from page 47. The dog, a long-haired, blind-man's dog, which had already served for one experiment, but whose health was completely re-established, as the meticulous chronicle informs us, received upon the forehead a blow delivered with violence enough, it was hoped, to kill it. The hope was not ill-founded; the animal remained comatose for thirty-six hours and then died. As the case is also a striking example of the general phenomena ascribed by the author to cerebro-spinal shock, a brief account of the symptoms may serve to show what he includes under that term. Before the experiment the dog's pulse was 100, respiration 24, and rectal temperature 38.8° centigrade. The blow, which was given at 9 A. M., was followed instantly by complete loss of consciousness, rigidity of the muscles, flow of blood from the nostrils, and involuntary defecation. During the first two minutes there were but three respirations and thirty pulsations. During the 4th minute four deep, difficult, stertorous respirations; pulse slow (12) and full; slight agitation of the limbs and tail; the respiration grew more rapid until the 8th minute, after which it remained at 32 until about the 25th minute when it was observed to have quickened to 42 with occasional arrests lasting for from 15 to 30 seconds. The pulse was 70, full and strong, each inspiration being marked by from two to four beats more feeble and rapid than the others. The thermometer placed in the rectum marked at the 6th minute 39.8°, at the 12th 39.2°, at the 25th 38.6°, and at the 60th 37.6°. The rigidity of the limbs, interrupted by occasional twitchings, especially of the muscles supplied by the facial nerve, gradually diminished and gave place to complete resolution during the second quarter of an hour. The respiration continued difficult and stertorous during the day, varying between 16 and 24; the pulse (including the rapid beats during each inspiration) was 80–90. The temperature at 5 P. M. was 34.2°. The next morning the animal was still unconscious; respiration 32, stertorous, pulse 90–100; temperature 38.8°. Resolution, pupils punctiform, sensibility obtuse, but reflex sensibility preserved He remained in this condition during the day and was found dead the next morning. The autopsy disclosed a fracture on each side beginning above the orbital arch, extending through it to the temporal fossa, and ending on the left side in the centre of that fossa, but curving inwards on the right towards the sella turcica. On the left was a small lenticular clot between the dura mater and the bone; the cavity of the arachnoid over the convexity of the right hemisphere contained a layer of blood not more than 1 millimetre in thickness. Both hemispheres were greatly congested throughout, "evidently the first stage of a generalized encephalitis following concussion." Ecchymoses in the sulci between the convolutions, and under the pia mater on each sphenoidal lobe. The ventricles were filled with reddish serum; there were five or six small hemorrhagic spots in the upper third of the floor of the fourth ventricle, the medulla oblongata was greatly congested and softened, and its surface of section showed minute hemorrhagic spots each surrounded by a zone of softening. The central canal of the cervical portion of the spinal cord was dilated with softening and punctiform hemorrhages in the gray substance of its walls.

We must distinguish between the early and late lesions, those which are the immediate consequence of the blow, and those which are due to the subsequent inflammation. Among the first are the extravasations of blood on the surface and in the bulb; it is plain that the latter organ was affected by the shock although the blow fell upon the forehead; and the dilatation of the central canal, corresponding to that observed in the first experiment mentioned, may be taken as a proof that in this case also the contents of the lateral ventricles were driven violently into the fourth ventricle, and were one at least of the causes of the extravasations found at the upper portion of its floor. Repetitions of the experiment, with blows of different force, and falling upon different regions of the skull, were made by M. Duret with objects and results which we must here pass by without description, but from which we may draw one inference not drawn by him. The rupture of vessels on the outside of the bulb, and the greater facility with which the characteristic lesions of the fourth ventricle were produced when the blow fell upon the forehead or even the occiput than when it fell upon the side of the head, would indicate some other mechanism than simple compression of the hemispheres forcing out the contents of the ventricles. A portion of this objection has not escaped his notice, and he seeks to explain the less effect of blows in the transverse axis by the action of a *cône de soulèvement*, an elevation of the skull produced by its elasticity at the point immediately opposite that at which the blow was received, and corresponding to the *cône de dépression* at that point. He thinks that the sort of vacuum thus formed solicits the liquid contained in the subarachnoid space and thus diminishes the effect of the cône de dépression. When a blow is delivered upon the vertex no cône de soulèvement can be formed because the opposite point rests upon the spine and cannot yield. It may be that all this is true, but there is nothing in the book to prove it, and it is not easy to believe without a demonstration that the elasticity of a globular body is manifested only in a change of form at the point opposite that at which the original impulse is received. As for the blow upon the vertex, the assumed absence of the cone of elevation can hardly be accounted for by the reason given, for if it could not be produced by its own actual projection, it could by a relative projection, that is by the rebound of the rest of the skull, or, as any one who has squeezed an orange or a rubber ball must have observed, its absence would be compensated for by the increase of all the transverse diameters. It seems a simpler and more probable explanation, if we may hazard one, to infer that a more complete or uniform compression is produced by a blow upon the forehead, since it acts directly upon both sides, and that its effect is increased by the fact that the movement of the wave being in the axis of the blow it is augmented by the direct application to it of the force.

It is frequently noted in the autopsies that clots of considerable size were found on the outer surface of the medulla oblongata, and M. Duret explains these, as he does all others on the surface, by the action of the peripheral wave,[1] that occupying the subarachnoid space. This wave, he thinks, distends the meshes of the space and thereby ruptures the vessels, just as a hypodermic injection sometimes ruptures those of the subcutaneous cellular tissue. That this action is exerted by the wave is proved beyond a question by some of his experiments, but it seems as if the generalization had been carried a trifle too far, and that the author's legitimate pride in the force discovered by him had led him to overlook a not unimportant adjuvant. The disparity between the extent of the peri-bulbar lesions and others likewise produced at a certain distance from the point receiving the blow leads one to look at once for a special reason. No such reason is presented by the sub-arachnoid space, its meshes are no larger, its capacity for distension even less than elsewhere, there is no reason to think that the liquid has a special tendency to collect there, on the contrary the distensibility of the neighbouring occipito-atlantoid ligament seems to furnish a sort of local safety-valve. What then can be the reason? The attention that is not pre-occupied falls at once upon the gross anatomical differences, the stalk-like form of the medulla, its position in the axis of the blow and at the very outlet of the skull—all point to a reason less recondite than that contained in the waves of the cerebro-spinal liquid. The medulla oblongata seems to be so exposed to the receipt of direct violence that it might almost be used as an argument against the doctrine of the survival of the fittest. It must be noticed too that the extravasations are found upon its anterior surface, there where it rests upon the basilar process at the very point where the impulse of the direct force transmitted through the bones would be received.

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[1] We use the term *wave* merely as a convenient one for expressing the movement of the liquid from one point to another; it must be remembered that the movement, in the subarachnoid space at least, takes place through a multitude of small channels, and is therefore a collection of streams rather than a single wave.

But these are only objections of detail; they leave this main fact that lesions are produced by peripheral and ventricular waves of the cerebro-spinal liquid unshaken. We have yet to consider the manner in which the waves are produced and propagated. The first of the essential elements in this consideration is the elasticity of the skull. We use the term elasticity advisedly, for the results in all the experiments were obtained without the aid of depressed fractures. The elasticity of the skull scarcely needs a demonstration. Almost every one knows that an empty skull let fall upon the pavement rebounds to the hand that dropped it; but the measure of the elasticity, the extent to which it yields, and therefore the measure of the compression which the blow exerts, is not so well understood. A single experiment puts it in a very clear light. Félizet (p. 28) filled a skull with melted paraffine, cooled it, and let it fall from a height of 2½ feet. The globe of paraffine was then removed and showed at the point corresponding to that which received the blow a flat circular surface more than an inch in diameter, and representing a depression of one-third of an inch, or in cubic measure .13 of a cubic inch, a very considerable amount for so moderate a blow. To understand its effects upon the contents of the skull we must turn to M. Duret's description of the disposition of the cerebro-spinal liquid.

The cerebro-spinal liquid is collected in two irregular masses, the peripheral and the ventricular, communicating with each other by a narrow opening at the lower end of the fourth ventricle, the foramen of Magendie. The arrangement of the ventricular portion is comparatively simple; it is contained within the ventricles and the central canal of the cord, and its different portions communicate quite freely with one another. In order to understand the action of this portion in producing the lesions of cerebro-spinal shock we must bear in mind the fact that the great part of it occupies the lateral and third ventricles, and communicates with the smaller part in the fourth ventricle through the aqueduct of Sylvius or iter e tertio ad quartum ventriculum.

The disposition of the peripheral portion is much more complex; it occupies the subarachnoid space covering the brain and cord, a space which, beginning in or continuous with the so-called lymphatic sheaths of the fine arterioles distributed to the substance of the brain, the *gaînes de Robin*, expands into larger channels in the sulci between the convolutions, and finally into sacs of different sizes at the sides and base of the brain. It is continuous also with the sheaths of the vessels and nerves of the organs of special sense. This arrangement is happily compared by M. Duret to a system of brooks, rivers, and lakes, the principal rivers being the channels in the fissures of Sylvius and Rolando, and the lakes the *sylvian* at the lower extremity of the fissure of Sylvius, the *central* at the base of the brain, the *inferior cerebellar* behind and below the cerebellum, and the *terminal* at the lower end of the spinal cord. The communication with the ventricular portion is through the foramen of Magendie, between the inferior cerebellar lake and the fourth ventricle.

The liquid is produced by exudation from the vessels, and its tension is greater than that of the air, being equal, according to Leyden, to that of a column of water 10 centimetres high, and rising during violent respiration to 14 or 15 centimetres. Of the many interesting points connected with the functions of this liquid we have space to mention only one, namely, the manner in which it protects the brain from injury by changes in the amount of blood supplied to it. It is apparent that as the cavity of the cranium is always completely filled, and its walls are not distensible, the brain would be compressed at each beat of the heart by the additional amount of blood forced into it, unless provision were made for a simultaneous withdrawal of a portion of the contents of the cranium. This provision is found in the veins and in the distensibility of a portion of the walls of the spinal canal. The cerebro-spinal liquid receives the impulse of the pulsation, gives way before the dilating arteries, presses upon and compresses the veins, and distends the membranous portions of the spinal canal. This can be clearly demonstrated by the graphic method at either end of the canal. If the occipito-atlantoid membrane is exposed by division of the muscles of the back of the neck, traces can be obtained showing the effect upon it of every pulsation and respiration.

As the sub-arachnoid space is not an empty sac like the cavity of the arachnoid, but is generally occupied by a delicate fibrous reticulum supporting the bloodvessels, in the meshes or intervals of which the liquid is contained, every sudden or extreme dilatation is attended by the risk of rupture of the filaments and vessels, and of consequent extravasation of blood. The same effect is produced, although by a different mechanism, in the minute prolongations of the cavity along the arterioles; when the peri-vascular sheaths are distended by the afflux of the liquid under the impulse communicated by a blow, the arterioles are compressed directly by the liquid, and the capillaries indirectly by pressure through the brain substance; then if the pressure is suddenly released the vessels burst by the unopposed tension of their contents. This has no special pathological significance, but it was very prettily shown by an experiment devised with this object. Pressure was gradually made upon the brain by an injection of wax, and maintained until the death of the animal; very few and slight hemorrhagic legions were found. The experiment was then repeated, and when the same degree of pressure had been obtained it was suddenly diminished; the tissue of the brain was then found to be filled with small hemorrhagic foci.

The effect of this pressure upon the respiration, and the dependence of the respiratory phenomena upon the cerebro-spinal liquid, are clearly shown by the experiment recorded on page 79. After section of the muscle of the back of the neck, and exposure of the occipito-atlantoid membrane, an injection of 15–20 grammes of wax was made rapidly through an opening in the anterior portion of the vertex. This was followed immediately by very violent opisthotonos and total arrest of respiration. After the animal had remained a minute and a half in this condition the bulging distended occipito-atlantoid membrane was punctured with a small triangular needle, and the liquid allowed to flow out; the animal immediately began to breathe again at the rate of sixteen respirations per minute. Two or three minutes later the respiration again ceased, and it was observed that the liquid was no longer flowing, and that the membrane was again bulging; a second puncture was followed by a second renewal of respiration; then, after three or four minutes a third arrest, third puncture, and permanent re-establishment of the breathing.

As the cerebro-spinal liquid is distributed so widely it is easy to understand that blows acting upon and through it should produce lesions at every part of the brain, although their severity at the different points would vary according to the facility with which the liquid could pass from one part of the sub-arachnoid space to another. While it is true that, in accordance with the well-known hydrostatic law, pressure upon an inclosed body of water will be felt equally at every point on the inclosing surface, the character and size of the channels of communications between the different parts of that space, if its walls are at all distensible, will modify the effects of that pressure by affecting the facility with which the liquid passes from one part to another.

This fact, while referred to indefinitely by M. Duret at one or two places in his book, does not receive from him the attention which it deserves, for by it alone can, among other things, the bursting pressure exerted within the fourth ventricle be explained. As that cavity communicates, through the foramen of Magendie, with the neighbouring sub-arachnoid space, the pressure within and without the ventricle when the liquid is at rest is the same, and the only effect of increase of that pressure would be increased compression of the bulb between the two bodies of liquid, as a piece of iron is compressed between the hammer and the anvil. But, as the communication through the foramen is insufficient to accommodate a wave of liquid (as shown by the rupture of its walls), and as similar obstacles prevent the timely arrival at the posterior cerebellar lake of the peripheral wave originating at the cone of depression, the unbalanced pressure within the ventricle produces rupture of its wall, just as a rubber ball is burst by a too vigorous attempt to force its contents through a small orifice. The distensibility of portions of the wall of the spinal canal affords a space into which the liquid contents of the cranium can pass, and the conditions of the problem, therefore, are not those of simple pressure, but the much more complex ones of liquids in motion; and although the effects of variation in pressure are shown in some of the lesions, the most important ones are those produced by the waves.

The lesions found on the convexity of the hemispheres are hemorrhages of greater or less extent from the vessels of the meninges, and, when the bone has been broken, of direct laceration. M. Duret thinks that when the bone has not been broken, the lesions found under the point where the blow was received are the result not of direct violence, but of the sudden springing back of the corresponding portion of bone after it has been depressed; into the vacuum thus formed the return wave rushes, and tears up the meningeal vessels. He rejects also the old explanation by contrecoup of the lesions at the opposite side of the skull, and substitutes one similar to that just mentioned, the vacuum being formed by an elevation of the bone corresponding to the depression produced at the opposite point by the blow (*cône de soulèvement*). We have already remarked that there is no proof of the existence of such an elevation, and, therefore, the argument based upon it cannot be accepted. The hemorrhages are in the form of ecchymoses under the pia mater, staining of the liquid in the sulci by rupture of the small vessels in the arachnoid meshes, and in small miliary foci due to rupture of the capillaries by changes of pressure in the lymphatic sheaths.

At the base of the brain the lesions are frequent and extensive, consisting of hemorrhages into the central and sylvian lakes, and sometimes, by rupture of its visceral layer, into the cavity of the arachnoid. Similar lesions are sometimes found around the bulb, and to a greater or less distance down the spinal cord.

The lesions within the fourth ventricle have already been described in part; they comprise extravasations of blood of greater or less size, most frequent at the lower angle, and next at the upper angle of the ventricle. These are sometimes extensive enough to give rise to the formation of a distinct clot, but most commonly are only miliary foci. It is worthy of note that a V-shaped mass of gray matter lying near the lower angle, and thought to be the origin of the pneumo-gastric nerve was frequently occupied by some of these foci. The foramen of Magendie is frequently torn, and in very severe cases the floor of the ventricle may be split. Interstitial hemorrhages are often found in the substance of the medulla and pons.

Finally, hemorrhagic lesions are sometimes produced at different points in the spinal cord. The case reported on page 102 is an interesting example of traumatic locomotor ataxy caused by a blow on the side of the head, the lesion consisting of an extravasation under the pia mater covering the posterior columns of the cord at the brachial enlargement.

The *symptoms* produced by cerebro-spinal shock are manifested in the muscles, respiration, circulation, and intelligence. The shock is followed instantly by an intense spasm and rigidity of the muscles, a spasm which is so general that it must be looked upon as reflex, as a manifestation of an irritation of sensitive centres, of the restiform bodies, according to M. Duret. This rigidity disappears in the course of a few minutes, and is followed, according to the severity of the shock, either by a return to the normal condition, or by complete resolution lasting until death. Agitation or paralysis of different groups of muscles is an indication of local lesions to be referred to hereafter.

With this spasm of the muscles of relation is associated a similar condition of the muscles of organic life, notably those of the arteries. These vessels contract, the tension of the blood contained in them is increased, and as a consequence of the obstruction which this offers to the passage of the blood through them, the tension in the veins is greatly diminished. In an experiment upon a horse the tension in the jugular vein sank from 16 centimetres to 4 at the moment of the shock, an enormous difference, which plainly shows how complete the anæmia of the brain must have been. This spasm is followed by a general dilatation of the vessels, the result either of fatigue or of inflammatory action set up by the numerous hemorrhagic lesions. The effect of these variations in the blood supply is seen in the functional disturbances of the brain; the primary anæmia causes complete loss of consciousness, which is prolonged by the succeeding hyperæmia, and may last, if a generalized inflammatory process is set up, until death.

We cannot follow the author through his examination of the many interesting consequences and incidents of this spasm, but will mention only the changes produced in the temperature which are ascribed by him to it. Starting with the theory of the profound antagonism between the vessels of the peripheral and of the visceral circulation, as established by the researches of Claude Bernard and other physiologists, he claims that, while there is arterial contraction in the brain and on the surface, there is arterial and capillary dilatation in the abdominal viscera, and this dilatation is marked by local rise of temperature. The facts are that the rectal temperature rises to 39.5° or 40° centigrade immediately after the shock, or even to 41° or 42° in very severe cases. It subsides promptly, falls below the normal, and remains there until death or the beginning of the inflammatory stage. This theory of visceral dilatation does not harmonize however with the preceding theory of generalized spasm ; and as we know that muscular action will raise the temperature of the body, and paralysis will lower it, it seems more reasonable to ascribe the rise of temperature to the intense action of the muscles, and its fall to the subsequent resolution which amounts practically to paralysis.

The contractions of the heart are at first precipitate, feeble, and incomplete, and soon become natural if the blow has been light. But if the blow is violent the pulse becomes slow, strong, and tense ; this corresponds to the period of contraction of the arteries. If the blow is still more violent the period is marked by a dicrotism during the inspiratory acts that may be mistaken for rapid and feeble pulsations. When dilatation succeeds the contraction of the arteries the pulse remains slow, but is soft instead of tense ; and, if finally the inflammatory stage is reached, the pulse becomes rapid.

The changes in respiration present five stages. The 1st is that of spasmodic arrest, and is common to both slight and severe shocks. It lasts only two or three minutes, and is followed by the stage of primitive acceleration which lasts fifteen or twenty minutes, and is the result of direct irritation of the respiratory centres. The 3d stage, that of irregularity and intermittence, is found only after severe shocks, and is due to lesion of the pneumogastric nerve itself, or of its origin in the gray matter at the lower angle of the fourth ventricle. The 4th stage, that of slowing, is marked by fulness and slowness of inspiration, followed without any interval by short expiration. The 5th stage is that of secondary inflammatory acceleration. Stertor is common in the severe cases, and is ascribed to traumatic paralysis of the muscles of the soft palate. In some of the recorded experiments it appears, however, to have been only an incident of the general muscular resolution ; and in two, pages 109 and 130, it existed only during short periods of insensibility.

Two experiments were made with the view to determine the points in the 4th ventricle, irritation of which would excite spasms of certain muscles, such as had been observed in some cases, especially those of the eyes, lips, and jaws. The results with reference to these points were in the main confirmatory of those obtained by other physiologists, but incidentally others were obtained which strikingly corroborate the author's explanation of the relations between the lesions and symptoms observed in the other experiments. The occipito-atlantoid membrane of a dog was exposed, a director passed upwards through it, and its point pressed against different parts of the floor of the ventricle. Not only was the expected action of the different muscles excited, but general rigidity, opisthotonos, arrest of respiration, unconsciousness, and loss of reflex sensibility were also produced. These experiments may be considered a positive proof of the causative relation existing between the lesions and symptoms observed in the other cases, and the previous experiments are an equally positive proof of the agency of the cerebro-spinal liquid in the production of the lesions in these other cases. The experiments of M. Duret have made it necessary for us to revise our opinions upon many cerebral traumatisms, and he deserves great credit for having discovered and demonstrated a factor in their production which has hitherto been overlooked.

The capital point in this discovery is the fact of the passage of the contents of the lateral ventricles into the fourth ventricle under the influence of pressure exerted upon the hemispheres, and this fact is to be taken into account in apoplexies as well as in traumatisms, for, as is shown in another part of the book, the injection of a small amount, 3 to 5 grammes, of a non-absorbable liquid into the substance of the brain or upon its surface is sufficient to produce the symptoms and lesions of cerebro-spinal shock; and an analysis of the symptoms of apoplexy shows a remarkable similarity, if not an absolute identity, with those above described. There is the same loss of consciousness, and its explanation by vascular spasm followed by dilatation is supported by post-mortem evidence. There is also the short convulsive stage (irritation of the sensitive tracts of the bulb), followed by complete resolution, with loss of both absolute and reflex sensibility. The pulse shows the same changes, at first slow and filiform, then natural but weak, then accelerated; and the analogy between the respiratory phenomena is particularly close. Finally, the temperature is also lowered, and rises again, of course, if the inflammatory stage is reached, and probably would show the short primitive rise corresponding to the spasm of the muscles if the observation could be made. Its short duration, ending perhaps before the arrival of the physician upon the scene, and the necessity of measuring it in the rectum are sufficient reasons for its having hitherto escaped observation. M. Duret claims that the mechanism by which these symptoms are produced is the same in both cases, namely, the sudden passage of the contents of the lateral ventricles into the fourth ventricle and the consequent irritation of the restiform bodies; and he intimates in a foot-note that this theory has received the support of Prof. Charcot. The statement, however, certainly needs to be accompanied by a limitation, one which doubtless was not overlooked by him, but is not expressed in set terms. The explanation can be true only of rapid hemorrhage from a vessel of considerable size, for the compression exerted by a slight hemorrhage, or by one occurring slowly, would be insufficient in amount or in rapidity of production to exert the mechanical action essential to this theory. This absence of the lesions and symptoms of cerebro-spinal shock, when the compression is increased slowly and gradually, is plainly shown in the histories of some of the experiments recorded in the chapter devoted to the examination of the mechanism and influence of compression.

It does not seem desirable to enter as fully into the author's examination of the effects of compression of the brain as into that of the cerebro-spinal shock, and we shall therefore confine ourselves in what follows to the more prominent results of this examination, leaving the others to be considered when the second or clinical part of the book shall have appeared.

It follows, of course, from what we have already learned of the cerebro-spinal liquid, that any compression of the brain, any diminution of the capacity of the cavity of the cranium, exerts an effect at all points occupied by that liquid, an effect dependent upon the degree of increase of its tension. This increased tension diminishes the calibre of the vessels (by pressure upon them) and the amount of blood which passes through them in a given time, and the results of this local anæmia are shown in the disturbance of the functions of the cerebrum and medulla. If the tension of the cerebro-spinal liquid becomes equal to that of the arterial blood, the circulation is arrested and the individual dies. This equality of intra- and extra-vascular tension cannot, however, be produced by an escape of blood into the cavity, unless the vessel ruptured is a very large one; and, moreover, the vascular spasm produced by the irritation of the sensitive nerves or centres increases the arterial tension so much (from 8 centimetres of mercury to 22 or even 28), that an equal extra-vascular pressure is probably produced only in experiments, and never in diseases or traumatisms. This increase of tension is marked by notable slowing of the pulse, except when it is great enough to cause death promptly; in this latter case the pulse is accelerated, usually to such a degree that it cannot be counted. The other symptoms, which become more marked as the pressure increases, are somnolence, dulness of intellect, and diminished sensibility or complete coma, according to the pressure. This increase of tension cannot be estimated either clinically or post-mortem, and in its place we have to consider the size of the clot, and decide from that whether the symptoms observed during life were due to compression or to some other pathological influence.

With the view of determining the relations between increased tension and the amount of compression, M. Duret made a number of experiments, consisting in the injection of wax into the arachnoid cavity or between the dura mater and bone, and found that in the latter case a clot equal in size to one-twelfth of the capacity of the cranium would cause coma and death in a few hours, while in the former case the clot produced these results only when it became equal in size to one-sixth or even one-fifth of the capacity of the cranium. In attempting to utilize these facts in clinical observation, however, we must remember that they apply only to *early* symptoms and to cases in which there is no concomitant lesion of the nerve centres; for the cause of the later results is found in the subsequent inflammation and changes in the circulation.

The symptoms produced by pressure exerted by increased tension of the cerebro-spinal liquid may be usefully classified according to their origin in the hemispheres or in the medulla. Those pertaining to the former are muscular weakness and dulness of the intellect and sensibility, varying directly in intensity with the pressure. The medulla and spinal cord being the centres of reflex sensibility, pressure upon them is manifested by dulness or abolition of reflex sensibility, for which touching of the cornea is the best test, because that organ is under the control of the medulla and is the last to lose its sensibility, both because it is near its nerve centre and because the vascular supply of the medulla is such that it resists pressure longer than any other part. This resistance of the medulla to traumatic influences led Charcot, in imitation of Haller, to call it the *ultimum moriens* of the nerve centres, and its possession of this quality is amply justified by the importance of the functions over which it presides—circulation, respiration, and animal heat. As a rule, pressure causes no change in either of these three functions until after the tension of the cerebro-spinal liquid has become nearly equal to that within the arteries; then both pulse and respiration grow slow, and the temperature of the body falls. If the tension is increased until it equals or exceeds that of the arterial blood, the temperature falls still lower and the respirations grow less frequent; but the pulsations suddenly become so rapid that they cannot be counted, and the animal dies in a few moments. A positive sign of obstruction to the circulation of the medulla is furnished by the character of the respiration (slow and deep inspiration with more rapid expiration) and the occurrence of three or four rapid pulsations during each respiratory act. (M. Duret does not seem to be quite sure of this observation, for he speaks of it in other places as a "dicrotism which might be mistaken for rapid pulsations.") The predominance of symptoms referable to the medulla is characteristic of intra-ventricular pressure (extravasations). In contrast to this, pressure upon the anterior surface of the hemispheres, if moderate, quickens the respiration and pulse; if greater, it slows the respiration, and after a longer interval the pulse also.

We need not follow the author in the parallel he draws between the symptoms of cerebro-spinal shock and of compression; for, although interesting, it is necessarily incomplete and must await a fuller development in the clinical portion of the work which is to follow, and we must close our review of this portion with a consideration of the important symptoms produced by lesions of the dura mater. Their importance is due to the fact that this membrane is well supplied with sensitive nerves coming mainly from the fifth pair, the physiological action of which can be easily excited by traumatisms of the skull or by inflammatory processes within it. The manifestations of this action complicate the symptoms due directly to the primary lesion and render an accurate diagnosis difficult, because some of them are also symptomatic of lesions of the brain substance. Without entering into any of the details of the experiments, we may give briefly the conclusions arrived at. As the nerves are nerves of sensation, pain is, of course, a frequent symptom, and the others are those of reflex irritation manifested by spasms and contractures in the muscles, by modifications of the respiration and pulse, and by vaso-motor changes in the eyes and brain, with consequent somnolence or coma. The spasms and contractures of the voluntary muscles are situated sometimes upon the same side as the lesion, sometimes upon the opposite side; they have a tendency to become more or less generalized, and involve the neighbouring groups of muscles, and never have the localization and voluntary appearance of contractions induced by lesions of the cortex.

The diagnosis of lesions of the dura mater, their differentiation from those of the brain substance itself, has a positive and practical importance, because they may be due to a depressed fragment of the inner table of the skull or to a clot of blood between the dura and the bone, and therefore amenable to surgical treatment.

If we compare them with the symptoms of the lesions accompanying cerebro-spinal shock, we find that in the latter a slight lesion, *e. g.*, a superficial extravasation, gives rise at first to symptoms indicating exaggerated functional irritability of the corresponding part of the brain, that is, to localized muscular twitchings. If the lesion is more severe, if it is destructive, it causes paralysis; and in both cases, when the lesion involves the cortex, the symptoms appear on the opposite side of the body. Permanent contraction of different muscles appears only secondarily, after the lapse of one or more days. It is at first limited to the muscles corresponding to the injured nerve centre and to the neighbouring groups, but may become generalized and even affect both sides of the body.

The symptoms produced by lesions of the cortex in the primary stage are thus seen to be entirely distinct from those due to lesions of the dura mater; in the former they occupy the side of the body opposite to the lesion; in the latter they occupy the same side: paralysis may occur in the former but not in the latter; in the former, spasmodic muscular action is distinctly localized and has a sort of voluntary character, while in the latter it is generalized and involuntary in appearance.

The permanent contractions of the second stage are attributed by M. Duret to subsequent irritation of the dura mater, and are therefore not to be distinguished from those produced by lesion of the dura alone. There is, however, one variety, and an important one, of muscular contractions excited by injury to the substance in which the dura mater plays no part; the irritation is propagated directly from the seat of the lesion to the medullary centres by the aid of the white motor fibres of the hemispheres. In this case the symptoms are always those of epileptiform attacks beginning usually in the muscles which correspond to the brain centre first affected.

The two remaining chapters of the book are devoted to the part played by the meningeal vessels in cerebral traumatisms, and to a résumé of the relations between the lesions of the different parts of the encephalon and particular symptoms, including a few contributions to the study of the localization of motor centres and the propriety of surgical interference based upon them. There is nothing in these chapters that requires especial mention here, and we now leave the book with the conviction that it is well worth study by all, and that as a product of accurate experimentation and cautious inference, it is not only a valuable contribution to the surgery and physiology of the brain, but also gives the assurance that French science will find in the new ranks men not unworthy to take the place of those who have made her past and present illustrious.

L. A. S.

ART. XXIII.—*A Clinical History of the Medical and Surgical Diseases of Women.* By ROBERT BARNES, M.D., Lond., Censor of the Royal College of Physicians, Obstetric Physician and Lecturer on Obstetrics and Dis. of Women to St. George's Hospital, etc. Second American from the second and revised London edition. 8vo., pp. 784. Philadelphia: Henry C. Lea, 1878.

THE American profession will receive with great pleasure this second edition of Dr. Barnes's excellent book. Dr. Barnes, in his brief visit to the United States in our Centennial year, made many acquaintances, many friends, and they will be glad to have him address them again, even though it be by the printed page, and from the other side of the Atlantic. Moreover the book especially commends itself to American readers by its recognition of American works and workers in gynæcology, and by its graceful dedication to one whom the profession of this country hold in such high honor, Dr. Fordyce Barker.

The title page states that this edition is from the second and revised London edition, and Dr. Barnes, in his preface, refers to the revision as having been "conscientious." But the thoroughness of this revision—its